part of the 20th century children with Down's syndrome were tested for syphilis; it was considered that syphilitic infection in their parents through damage to the germ cells could have led to the production of offspring with this syndrome. I was even more interested to find, however, that in a couple of American studies[1 2] some positive Wassermann and Lange gold chloride test results were obtained and it was felt that it could be concluded "the tests prove beyond question that this condition [Down's syndrome] is a result of syphilitic infection".[2]

These were almost certainly false positive syphilis test results. Such results may occur in autoimmune disorders. In the Wassermann reaction, certain autoantibodies crossreact with cardiolipin, and it is, I think, relevant here that findings indicative of autoimmune haemolytic anaemia have been often found in Down's syndrome. Positive colloidal gold chloride test results may be obtained in immune-mediated neuropathies as a result of elevation of CSF protein, and pertinent findings in Down's syndrome include demyelination in brain and spinal cord, sensory and motor disturbances, and intellectual impairment.

Autoantibodies have been detected quite frequently in people with Down's syndrome as part of a study of their thyroid glands, and I believe that the possibility needs to be considered that a great many of the disorders of Down's syndrome are autoimmune in origin. Fibroblasts trisomic for chromosome 21 show a significantly enhanced response to the antiviral effects of both type I and type II interferon,[3] and IFN-gamma has been found to be a very good in vitro stimulus for the induction of HLA-DR expression in a number of endocrine tissues, including thyroid epithelium.[4] It has been hypothesised previously that IFN-gamma production by normal blood lymphocytes is at too low a concentration in vivo to allow 1a antigen expression by normal human thyroid cells.[5] It is possible that the increased sensitivity of Down's syndrome cells to type I and type II interferon could lead to thyroid cells (*and potentially many other cells*, as well as the endocrine) expressing autoantigens and, subsequently, autoimmune disease developing.

Thus, rather than Down's syndrome being a result of syphilis as postulated in 1916, I believe that it should be added to the list of disorders which, through an autoimmune mechanism, can give rise to biological false positive tests for syphilis.

LILIAN ZIHNI
6 Oakington Avenue, Wembley, Middlesex HA9 8JA, UK

1 Stevens H C. Mongolian idiocy and syphilis *JAMA* 1915; **64**:1638.
2 Stevens H C. The spinal fluid in Mongolian idiocy. *JAMA* 1916;**66**:1374.
3 Epstein CJ, Epstein LB. Gene dosage effects in trisomy 21. In: de la Cruz F, Gerard Park S, eds. *Trisomy 21. Research Perspectives*. Baltimore, University Park Press. 1981:237–60.
4 Todd I, Pujol-Borrell R, Hammond R, Bottazzo G, Feldmann M. Interferon-gamma induces HLA-DR expression in thyroid epithelium. *Clin Exp Immunol* 1985;**61**:265.
5 Weetman A P. "HLA-DR antigen expression and autoimmunity" In: *Immunology of Endocrine Disease*. McGregor AM, ed. Lancaster, MTP Press. 1986:152.

I thank the Wellcome Trust which provided me with a 1 year post-doctoral fellowship. This work was carried out at The Wellcome Institute for the History of the Medicine, London.

Accepted for publication 10 June 1993

Penicillinase producing *Neisseria gonorrhoeae* associated with severe vulvovaginitis in a post menopausal woman

I was interested to note the case report from Bodsworth *et al*[1] of gonococcal mastitis in a male and the detailed study of penicillinase producing *Neisseria gonorrhoeae* (PPNG) from Warren and Phillips.[2] I would like to report a further atypical presentation of gonococcal infection in which a PPNG was isolated.

A 54 year old patient attended the department of GenitoUrinary Medicine in Swansea in August 1992 following referral from her general practitioner. She complained of malodorous vaginal discharge, superficial dysuria and severe vulval soreness. Symptoms had started about 24 hours after a casual unprotected sexual encounter whilst on holiday on a Greek island some two weeks previously. She had had a hysterectomy for menorrhagia 10 years before and was not taking hormone replacement therapy. Previous intercourse had occurred over eighteen months prior to the single sexual encounter which led to her attendance.

On examination of the vulva, a putrid odour was apparent and the vulval mucosa was uniformly red, swollen and covered with sticky yellow secretions. The appearance and texture of the vulva was that of recently cut silverside of beef. No vulval or perianal fissures or urethral discharge were noted and on passing a vaginal speculum a copious viscous yellow discharge coated the vaginal walls. The vaginal mucosa was uniformly red but less swollen than the vulva. The cervix was absent. There was no inguinal lymphadenopathy and no systemic symptoms or signs of infection. High vaginal swabs were taken for microscopy and culture for *Neisseria gonorrhoea*, *Trichomonas vaginalis* and *Candida* species. An ELISA test (Novo Nordisk) for *Chlamydia trachomatis* was also performed on the vaginal secretions. Urethral swabs were taken for microscopy and culture of *N gonorrhoeae*.

Examination of a wet-mount preparation of vaginal secretions was negative for trichomonads and fungal elements but masses of polymorphonuclear leucocytes (PMNLs) were noted. A Gram-stained preparation of the secretions revealed confluent PNMLs the majority of which contained Gram negative intracellular diplococci morphologically identical to *Neisseria gonorrhoeae*. Scattered parabasal/intermediate cells and a scanty mixed anaerobic vaginosis type bacterial flora[3] was also noted. The urethral swab revealed occasional PNMLs but no intracellular diplococci. A provisional diagnosis of gonococcal infection associated with an

anaerobic vaginal flora was made. The patient was allergic to penicillin and was given ciprofloxacin 250mg as a single dose followed by metronidazole 400mg twice twice daily for 5 days and oxytetracycline 500mg four times a day for 10 days. Topical clotrimazole 1% / hydrocortisone 1% (Canesten-HC) was also prescribed for symptomatic relief and as prophylaxis in case of subsequent development of vaginal yeast infection. Cultures of the vaginal and urethral swabs were both positive for *Neiserria gonorrhoeae* (beta lactamase positive, auxotype NR, serotype IB 18, plasmids 2;6;4·4;24;5.) Culture of the vaginal secretions was negative for yeast infection and *Trichomonas vaginalis*. The ELISA test for chlamydia was also negative. The patient was seen for follow-up a month later when she was asymptomatic and clinically the vulval and vaginal swelling and erythema had resolved but a marked atrophic vulvo-vaginitis was now apparent. Gram staining for the vaginal secretions was typical of a poorly oestrogenised post-menopausal vaginal smear in that it showed persisting AV type bacterial flora and many parabasal cells were noted. The number of PMNLs were reduced and there were no Gram negative intracellular diplococci. Vaginal pH was 6·0 (Whatman pH paper narrow range pH 4–6). Repeat cultures for gonorrhoea (including on this occasion, rectal and throats swabs) were negative.

Gonococcal vulvo-vaginitis is a recognised condition in pre-pubertal females but I have been unable to find any previous reference to it occuring in an adult. This patient had had a hysterectomy thus excluding the cervix as the site of primary infection and although it is theoretically possible that the primary infection in this patient was that of gonococcal urethritis, clinically the main site of infection was very definitely the vagina. It has been suggested that in prepubertal females the thin vaginal mucosa and relatively alkaline vaginal pH permit colonisation by gonococci which may be acquired from fomites or sexual contact.[4,5] In contrast the well oestrogenised thick vaginal mucosa and acid secretions (probably associated with H_2O_2-producing lactobacilli) of the adult pre-menopausal female are thought to protect the vagina from infection leaving the relatively alkaline cervical columnar epithelial cells as the main site of infection. It is probable that low oestrogen levels and associated atrophy of the vulvo-vaginal mucosa, lack of H_2O_2-producing labctobacilli and relatively alkaline vaginal pH in post-menopausal woman may mimic the prepubertal state and predispose to gonococcal infection. The case presented was very unusual in that the combination of her post-menopausal state, previous hysterectomy, casual intercourse with a man infected with PPNG and attendance at a genito urinary clinic before antimicrobial treatment is a rare occurrence which may explain why I have found no similar case in the literature. Patients in this age group are often not perceived to be sexually active, at least not with casual partners and it is fortunate that this lady had an astute general practitioner who referred her before giving treatment. The patient made an interesting social comment when asked about her sexual encounter. Apparently in the particular holiday resort she visited, the local Greek men prefer middle-aged single, widowed or divorced women in that they are considered "safe" with regard to human immunodeficiency virus infection and possible attack from jealous partners. The case may indicate that gonococcal vulvo-vaginitis is not confined to prepubertal females and the social changes enlarge the group of patients who are at risk of sexually transmitted infection. Such patients should be warned of these risks and encouraged to take precautions if they have a casual sexual encounter.

AL BLACKWELL
Singleton Hospital,
Sketty, Swansea SA2 8QA, UK

1 Bodsworth NJ, Price R, Nelson M. A case of gonococcal mastitis in a male. *Genitourin Med* 1993;69:222–3.
2 Warren C, Phillips I. Penicillinase producing *Neisseria gonorrhoea* from St Thomas' Hospital 1976–1990. The first fifteen years. *Genitourin Med* 1993;69:201–7.
3 Blackwell AL, Barlow D. Clinical diagnosis of anaerobic vaginosis (non-specific vaginitis): a practical guide. *Br J Venereal Dis* 1982;58:387–93.
4 Litt IF, Edberg SC, Finberg L. Gonorrhoea in children and adolescents: a current review. *J Paediatrics* 1974; 85:595–607.
5 Neinstein L, Goldenring J, Carpenter S. Nonsexual transmission of sexually transmitted diseases: an infrequent occurrence. *Paediatrics* 1984;74:67–76.

Accepted for publication 29 July 1993.

Chronic perianal ulcerations: role of *Trichomonas vaginalis*?

Trichomonas vaginalis is a flagellated protozoan which causes a sexually transmitted disease (STD) that usually affects women. Clinical symptoms in women are frequent, whereas for men the infection is often asymptomatic.[1] Rectal ulcerations with rectovaginal fistula[2] and reactive arthritis[3] were observed in women with a trichomoniasis genital infection. In both cases, the antigen HLA-B 27 was positive. Recently, a 61-year-old, married, heterosexual man, presented with a 1 year history of chronic perianal ulcerations. There was no history of genital or oral involvement. Empirical local treatments with corticosteroids, anti-bacterial and antifungal creams were ineffective. He was on no regular medication and his general health was reasonably good.

Routine laboratory tests (virological, fungal and bacterial cultures from ulcers and faeces) were negative. The Diamond's medium cultures revealed *T vaginalis* and perianal biopsy showed a dense infiltrate of neutrophils in the upper dermis. Urethral cultures for *T vaginalis* were also positive whereas the patient exhibited no urogenital symptoms. Syphilis, HIV and chlamydial repeated serologies were negative as were amoebiasis, ascariasis and bilharziasis serologies. HLA-B 27 antigen was also negative. Panendoscopy of the digestive tract was normal. Genital examination of his spouse revealed an